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Induced moulting issues and alternative dietary strategies for the egg industry in the United States

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The United States (U.S.) poultry industry continues to implement induced moulting to extend egg production in commercial laying flocks. Achieving an optimal moult requires dietary manipulation to cause a complete regression of the reproductive organs and cessation of egg production. This is followed by rejuvenation and initiation of an additional egg laying cycle. Currently feed withdrawal is the primary means to initiate moult and is regarded as an optimal approach for achieving post-moult performance. However, removal of feed can lead to potential physiological stress in laying hens as well as an increased susceptibility to *Salmonella enteritidis* colonization and invasion. To retain the ecological benefits of induced moult will require development, testing and implementation of alternative dietary approaches that minimizes these problems and increase the egg production and egg quality benefits associated with the additional egg laying cycles. Strategies for accomplishing this are discussed.

Keywords: induced moulting; egg production; bone remodelling; alternative moulting diets

Abbreviation index: U.S. = United States; cAMP = cyclic adenosine monophosphate; LH = luteinizing hormone; FSH = follicle-stimulating hormone; GnRH = gonadotropin-releasing hormone; PRL = Prolactin; T_3 = triiodothyronine; T_4 = thyroxine; TRH = thyroid hormone-releasing hormone; VFS = visceral forebrain system; VIP = vasoactive intestinal polypeptide.

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Introduction

Induced moulting is an important management tool for the U.S. egg industry. In addition to the economic benefits associated with additional egg laying cycles, moulting provides the commercial egg producer flexibility in the management of a flock to respond rapidly to shifts in the egg market as well as changes in feed and other input costs. Farms using the single-cycle (non-moulted) programme would need approximately 8.4 new flocks per layer house over a 10 year period, whereas only 5.7 flocks would be required for a typical two-cycle (one-moult) flock system (Bell, 2003). Therefore, 47% more hens would be needed to maintain houses at full capacity with the one-cycle option (Bell, 2003). Induced moulting rejuvenates laying hens for a second or third cycle of production, resulting in higher egg production, heavier egg weight, and improvements in egg quality parameters, such as albumen height, shell thickness, and specific gravity (Len *et al.*, 1964; Lee, 1982; Baker *et al.*, 1983; Bell, 2003). However, despite the historical precedent for successful utilization of moulting, controversy has come to the forefront in attempts to halt induced moults as they are currently practiced in the United States. A recent scientific symposium was held to discuss some of the consequences associated with current practices as well as future directions and a scientific perspective basis for understanding induced moulting (Gast and Ricke, 2003). This review represents a summary of current issues and potential strategies for moult induction.

Natural and induced moulting: physiological aspects

Annually, many wild birds naturally experience a body weight of loss up to 40%, accompanied by feather loss and regression of the reproductive system (Brake and Thaxton, 1979; Mrosovsky and Sherry, 1980). Establishment of broodiness seems to be the primary initiating factor for natural moult (Berry, 2003). Avians experience reduction of feed intake and body weight during incubation and brooding (Sherry *et al.*, 1980; Ankney and MacInnes, 1978). These physiological changes cause the cessation of reproduction. Therefore, wild birds take a self-induced rest to rejuvenate body tissues and build up energy stores. Based on these characteristics, the practice of induced moulting by the U.S. poultry industry by light manipulation and reduced daily feed intake closely mimic what occurs naturally. The combination of feed removal and light reduction from 16 h of darkness to 8 h of light for 10 to 14 days is the most widely used practice for induce moulting in the U.S. commercial egg layer industry (Bell, 1987; Holt, 1993; Holt, 2003). Induced moulting allows an egg producer to achieve a second productive laying cycle (Holt, 1999; Bell and Weaver, 2001). Light stimulation results in the release of the follicle-stimulating hormone (FSH) from the pituitary, which is followed by an increase in the growth of the ovarian follicles. Once reaching maturity, the ovum is released by luteinizing hormone (LH).

The process of moulting and the subsequent recovery from the moult appear to be a complex physiological mechanism involving endocrine systems, reproductive tissue structure and function, lymphoid structure, and immune function (Berry, 2003). A general increase in reproductive performance results from the rejuvenation effect. This rejuvenation may be associated with an increased tissue sensitivity or efficiency and reorganization of metabolic processes (Brake and Thaxton, 1979). In addition, the loss of adipose tissue may be associated with the overall increase in performance (Brake and Thaxton, 1979). However, both the regression and redevelopment of organs and tissues are related to the increased reproductive performance post-moult. The decrease in body weight of hens by feed withdrawal is directly related to decreased muscle, adipose tissue,

liver, and the involution of reproductive organs (Brake and Thaxton, 1979; Berry and Brake, 1985). Approximately 25% of the body weight loss is connected to the decrease in liver weight and involution of the reproductive organs (Brake and Thaxton, 1979). The loss of primary flight feathers is also involved in post-moult reproductive performance (Andrews *et al.*, 1987a, 1987b; Herremans *et al.*, 1988). The loss of primary feathers is clearly due to the loss of oestrogenic influence on the feather papilla (Péczely, 1992). Therefore, moult occurs during the lowest point in oestrogen production.

Lee (1982) reported that moulted birds exhibit greater egg production, better feed efficiency, better shell quality, and less mortality than unmoulted birds. A greater primary feather loss is associated with greater egg production (Lee, 1982). The peaks of egg production during the second cycle are approximately 75 to 85% (Bell, 2003). One of the main reasons for increased post-moult egg production is decreased post-moult production of shell-less eggs (Roland and Brake, 1982). Hens that lay shell-less or poorly shelled eggs show increased shell gland lipid (Roland *et al.*, 1977). This lipid is largely confined to the calcium secreting glandular epithelium (Baker *et al.*, 1983) and remains during the feed withdrawal periods until more than 25% of the bird's initial weight is lost (Brake, 1992).

Changes in circulating populations of blood leucocytes can occur during moulting. Alodan and Mashaly (1999) reported that heterophil numbers increased during 10 and 14 day periods of feed withdrawal. Davis *et al.* (2000) and Medvedev *et al.* (2002) indicated that heterophil:lymphocyte ratios were increased during induced moulting. Changes in heterophil:lymphocyte ratios can occur by a psychogenic influence during feed withdrawal (Webster, 2003).

Induced moulting: endocrinology and cellular aspects

A main function for luteinizing hormone (LH) is to induce ovulation. Premature ovulation is stimulated following injection of LH (Imai, 1973). Other functions of LH are associated with steroidogenesis and reduction of plasminogen activator activity (Shahabi *et al.*, 1975; Etches and Cunningham, 1976). LH peaks 4 to 6 hr before the hen ovulates (Johnson and van Tienhoven, 1980). Some studies reported that LH has an additional peak 14-11 hr prior to ovulation (Etches and Cheng, 1981) although the significance of this second peak was not determined. Shahabi *et al.* (1975) reported that mammalian LH *in vivo* increases plasma concentrations of progesterone, oestrogen, and testosterone production of the hen's ovary. A main function for follicle-stimulating hormone (FSH) is closely related to granulosa cell differentiation and the induction of steroidogenesis in prehierarchal follicle granulosa cells (Etches, 1990). FSH stimulates both cyclic adenosine monophosphate (cAMP) formation and progesterone secretion by the granulosa of intermediate stage follicles. A rise in the plasma concentrations of follicle-stimulating hormone (FSH) is determined 15 hr prior to ovulation in the domestic hen (Scanes *et al.*, 1977). Progesterone can induce both a preovulatory surge of LH and premature ovulation. The highest plasma concentration of progesterone occurs 6 to 4 hr prior to ovulation and coincides with the pre-ovulatory LH peak (Etches, 1990). Although the main function of androgens is unclear, high circulating concentrations of testosterone are necessary to stimulate LH secretion and induce ovulation. Moreover, ovulation can occur in the absence of any preovulatory increase in plasma testosterone (Johnson and van Tienhoven, 1984).

Like testosterone, oestrogens are not directly related to the induction of LH secretion or ovulation (Etches, 1987). Moreover, ovulation can occur in the absence of a preovulatory increase in plasma oestrogens (Etches, 1987). However, oestrogens are associated with reproduction, including the regulation of calcium metabolism for shell formation (Etches, 1987), induction of progesterone receptor in the ovary and reproductive parts (Pageaux *et*

al., 1983). Corticosterone controls the timing of the preovulatory LH surge (Wilson and Cunningham, 1980), but there is no ovulation–related increase in circulating corticosterone. In many avian species, circulating prolactin increases at the onset of egg laying. Rozenboim *et al.* (1993) reported that prolactin can reduce hypothalamic GnRH levels and inhibit LH secretion. Berry (2003) indicated that these actions of prolactin would lead to involution of the ovary with reduced ovarian steroidogenesis and regression of the oviduct.

During the moulting periods, plasma concentrations of prolactin are less than half those determined in laying turkeys (Proudman and Opel, 1981). Circulating plasma concentrations of progesterone, LH, and oestradiol are lower in moulting laying hens than in unmoulting laying hens while corticosterone, thyroxine (T_4) and tri-iodothyronine (T_3) levels increase during the moult (Hoshino *et al.*, 1988). Hoshino *et al.* (1988) indicated that the declines of LH, oestradiol, and progesterone were coincident with the cessation of egg production. Verheyen *et al.* (1987) reported that the response of plasma progesterone to LH in moulting hens was reduced or delayed after LH injection. Thyroid hormones are elevated in blood plasma during the loss and regeneration of feathers (Brake *et al.*, 1979; Lien and Siopes, 1989).

Several studies have evaluated the effects of administrating thyroxine (T_4), prolactin (PRL), and gonadotropin-releasing hormone (GnRH) agonist on moulting to establish the relationship between hormones/neurotransmitters and moulting (Juhn and Harris, 1956; Decuypere and Verheyen, 1986; Dickerman and Bahr, 1989; Reinert and Wilson, 1997). T_4 treatment enhanced acid phosphatase activity in feather tracts (Kobayashi *et al.*, 1955), indicating a direct effect of T_4 on the feather papillae (Tanabe *et al.*, 1957). Medium and high doses of T_4 treatment groups without feed withdrawal displayed postnuptial moult (Reinert and Wilson, 1997). In addition, GnRH agonists cause ovarian regression through a down regulation of pituitary GnRH receptors (Dickerman and Bahr, 1989). GnRH agonists significantly reduced prostaglandin before the beginning of moult, suggesting marked ovarian regression (Kuenzel, 2003). PRL-treated hens also exhibited a rapid onset of moult with the least impact on laying behaviour (Juhn and Harris, 1956). PRL administration caused moult in castrated cockerels (Juhn and Harris, 1958). These hormones and neuropeptides related to the neuroendocrine system have potential to replace the conventional feed withdrawal programs. Since feed withdrawal methods have received tremendous negative attention related to the animal welfare issue in recent years (Bar *et al.*, 2003; Gast and Ricke, 2003; Webster, 2003), induced moulting using the administration of these hormones and neuropeptides could help reduce hen physiological stress during moulting.

It is well-known that induced moulting improves eggshell quality and egg production (Berry and Baker, 1991; Baker *et al.*, 1993; Hurwitz *et al.*, 1998). Heryanto *et al.* (1997) suggests that the regression and removal of the old tissues and the recovery of the tissues with proliferation and cytodifferentiation of new cells may improve post-moulting egg production and egg quality. In particular, rejuvenation of shell gland tissue may increase calcium-binding protein (Calbindin), resulting in improvement of eggshell quality (Heryanto *et al.*, 1997). The mechanism for Ca^{2+} transport to the eggshell is related to a vitamin D-dependent Ca^{2+} absorption and a multifactor-dependent transfer of Ca^{2+} to the shell (Yosefi *et al.*, 2003). Both steps are mediated by calbindin, which is found in both intestine and eggshell gland (Berry and Brake, 1991; Bar *et al.*, 1999). Calbindin increases with the onset of egg production and decreases as egg production reduces (Nys *et al.*, 1989). The relationship between eggshell thickness and the shell gland calbindin is positively correlated (Nys *et al.*, 1986). Berry and Brake (1991) reported that moulting treatments significantly increased the amount of calbindin in the shell gland and duodenum of moulted hens compared to non-moulted hens. However, recently Yesefi *et*

al. (2003) reported that calbindin levels in duodenal and shell gland in the moulted hens were not significantly different compared to those in the non-moulted hens, suggesting the improvement of shell quality in the moulted hens does not involve mechanisms associated with calbindin system. Thus, additional and/or other mechanisms may be involved in improvement of eggshell quality.

Bone remodelling during moulting and potential strategy for improving bone strength and skeletal integrity after moulting

Structural bone loss during the laying period can cause fragility and susceptibility to fracture (Whitehead and Fleming, 2000). Structural bone loss in laying hens is an important animal welfare and economic issue for poultry industry. Several studies have reported that approximately 30% of hens housed in batteries suffer at least one broken bone during their lifetime (Gregory and Wilkins, 1989). Approximately one-third of these occur while housed in the cages with the remaining occurring during depopulation, transport, and processing. Induced moulting by feed removal is a potential factor escalating structural bone loss in old laying hens. Several studies have indicated that feed removal greatly influences bone qualities in laying hens. Garlich *et al.* (1984) reported that the induced moulting using feed removal significantly reduced femur weight and density of laying hens. Recently, Mazzuco *et al.* (2003) confirmed that moulted hens by fasting for 10 days had a precipitous decrease in bone mineral densities compared to non-moulted hens. They also indicate that bone minerals are slowly restored after moulted hens are fed standard layer rations. However, Newman and Leeson (1999) indicated that bone strength did not return to pre-fast levels although bone strength increased when moulted hens were fed a standard layer ration. Thus, the role of post-moulting layer rations is critical in order to maximize bone mineral deposition and improve skeletal integrity after moulting.

Fluoride is one of the candidates to stimulate bone mineral restoration and improve skeletal integrity after moulting. Fluoride has been one of the most successful agents used for enhancing bone mass in the treatment of osteoporosis (Rodríguez and Rosselot, 2001). Many studies have shown that fluoride has an anabolic effect on bone formation (Tenenbaum *et al.*, 1991; Marie *et al.*, 1992; Modrowski *et al.*, 1992; Chavassieux *et al.*, 1993; Kassem *et al.*, 1994). Takada *et al.* (1996) demonstrated that fluoride treatment increased the number of osteoblast (bone forming cells), indicating that the osteogenic effect of fluoride is mediated by changes in osteoblasts proliferation rate. Furthermore, increases in the number of active osteoblasts, the matrix apposition rate, and the mineral apposition rate by fluoride treatments have been reported (Ream, 1981; Marie and Hott, 1986). Grynpass (1993) has also demonstrated that fluoride treatment has an effect on bone mineralization by increasing calcium phosphate deposition. Various animal models have been used to research the impacts of fluoride on bone tissue. In ovariectomized rats, fluoride treatment markedly increased and restored bone calcium content to normal rats (Modrowski *et al.*, 1992). Several researchers have reported that fluoride treatments improved skeletal quality in the poultry. Merkley (1976) indicated that fluoride supplementation into water significantly increased bone strength in cage-reared broilers, in which bone breakage during processing is a considerable problem. Merkley (1981) also demonstrated that fluoride treatment increased the breaking strength of humeri from 6.86 to 13.35 kg and that of tibiae from 6.61 to 13.10 kg in caged laying hens. The fluoride treatment also significantly increased the percentage of bone ash. Rennie *et al.* (1997) showed that 200 ppm of sodium fluoride supplementation into a layer ration stimulated medullary formation, increasing medullary bone volume.

Current issues on induced moulting by feed withdrawal: physiology and behaviour

The practice of feed withdrawal is an efficient method to induce a moult because it is management friendly, economically advantageous and results in satisfactory post-moult performance for the commercial layer industry (Brake, 1993). However, a long period of feed withdrawal is more controversial. Poultry scientists, commercial poultry laymen and others are becoming more exposed to issues related to animal welfare occurring during induced moulting (Gast and Ricke, 2003). Although birds experiencing a natural moult do reject feed for a prolonged period of time (Mrosovsky and Sherry, 1980), the animal welfare concern is focused on whether it is detrimental to initiate moult before the bird is physiologically ready (Ruszler, 1998). Hens respond to long-term feed removal in three phases as summarized by Webster (2003): 1) Phase 1 lasts a few days during which physiological and behavioural adjustments reduce protein catabolism and energy expenditure, 2) During Phase 2, proteins are spared and lipids are catabolized to supply energy, and 3) During Phase 3, protein catabolism accelerates, and the bird halts visible activity and no longer eats. During fasting, aggression by the dominant hens against the subordinate hen can be highly elevated (Duncan and Wood-Gush, 1971). Fasting hens also will tend to exhibit increased behavioural activation such as increased standing, head movement, and non-nutritive pecking (Webster, 2003).

A neural pathway may be involved in regulating moult (Kuenzel, 2003). Several hormonal and neuropeptide treatments, such as thyroxine, progesterone, gonadotropin-releasing hormone agonist, and prolactin, can influence moult. In particular, thyroxine and prolactin administration can have considerable impact on moulting (Juhn and Harris, 1958; Reinert and Wilson, 1997). Thyroid hormone releasing hormone (TRH) and vasoactive intestinal polypeptide (VIP) are the neural modulators to release thyroxine and prolactin, respectively (Kuenzel, 2003). VIP-containing neurons and fibres are a neural system in birds comparable to the visceral forebrain system (VFS) in mammals, which regulates the balance of the autonomic nervous system (Kuenzel, 2003). Thus, VIP may have an important function to shut down the reproductive system by starting incubation behaviour followed by postnuptial moulting (Kuenzel, 2003). TRH has effects on moult when TRH-containing neurons form a terminal field in the external zone of the median eminence (Józra *et al.*, 1988). TRH changes the balance of the autonomic nervous system in the direction of the sympathetic nervous system to complete postnuptial moult (Kuenzel, 2003).

Current issues on induced moulting by feed withdrawal: susceptibility to feedborne *Salmonella* infection

Birds moulted by feed withdrawal may be more susceptible to *Salmonella enterica* subspecies *enterica* serova *enteritidis (S. enteritidis)* infection because induced moulting by feed withdrawal depresses the cellular immune response (Holt, 1992; Holt and Porter, 1992a, 1992b; Holt, 1993; Holt *et al.*, 1994; Holt *et al.*, 1995). The increased susceptibility to *S. enteritidis* infections in moulted birds has been connected to a rapid and severe inflammatory response in caeca, which was not generally seen in non-moulted infected birds (Isobe and Lillehoj, 1992; Porter and Holt, 1993; Macri *et al.*, 1997). During the moulting periods, *S. enteritidis* is readily transmitted to uninfected birds from infected birds in adjacent cages (Holt and Porter, 1992b), and can be transmitted to uninfected birds in cages a distance away from the infected birds, suggesting that airborne transmission of the organism may be important (Holt *et al.*, 1998). Moulted hens excreted significantly

higher *S. enteritidis* numbers in the faeces and possess greater levels of *S. enteritidis* in the internal organs (Holt, 2003).

S. enteritidis colonizes the gastrointestinal tract in birds prior to dissemination to multiple organs (Gast and Beard, 1993; Gast, 1994). The caecum is the main site for colonization by *S. enteritidis*, followed by the colon and ileum (Fanelli *et al.*, 1971; Turnbull and Snoeyenbos, 1974; Holt *et al.*, 1995). Colonization of the ovary may cause the production of *S. enteritidis* contaminated eggs, which in turn could lead to human salmonellosis if infected eggs are mishandled or undercooked. Therefore, the large numbers of *S. enteritidis* potentially shed into the house environment during moulting, may pose a greater problem for the producer and for future flocks that would occupy that house.

Effects of feed withdrawal induced moulting on the microenvironment of crop and caeca

Induced moulting through feed withdrawal alters the microenvironment of crop and caeca which are the main sites of *Salmonella* colonization in the chicken intestine (Brownell *et al.*, 1970; Soerjadi *et al.*, 1981; Impey and Mead, 1989, Durant *et al.*, 1999, Ricke, 2003). Corrier *et al.* (1997) reported that induced moulting had no apparent effect on pH or on the oxidation-reduction potential of the caeca, but induced moulting decreased in concentrations of acetic, propionic, and total volatile fatty acids (VFA) of the caeca. Corrier *et al.* (1997) suggested that increased susceptibility of moulted hens to *S. enteritidis* colonization may be related to decreased fermentation and production of VFA-producing bacteria present in the caeca, and addition of lactose in the drinking water during the water may enhance resistance to *S. enteritidis* colonization. VFA in the caecal contents in chicken are fermentation products of normal indigenous anaerobic bacteria (Barnes *et al.*, 1979, 1980) and the concentrations of VFA may represent the degree of fermentation activity of bacteria in the caeca (Barnes *et al.*, 1980).

Durant *et al.* (1999) reported that induced moulting increased crop pH, and decreased the *Lactobacilli* populations and the concentrations of lactic, acetic, propionic, butyric, and VFA of the crop. They also reported that increased *S.* Enteritidis colonization in the crop was due to the high pH and lowered concentrations of lactate and total VFA. *Lactobacilli* that are the predominant microflora in the crop, play an important role in maintaining a low pH that prevented coliform establishment in the crop (Fuller and Brooker, 1974) and in preventing the growth of *E. coli in vitro* (Fuller, 1977). The native intestinal microflora functions as a protector against *Salmonella* colonization of the caeca (Nurmi and Rantala, 1973; Barnes *at al.*, 1980; Nisbet *et al.*, 1994; Corrier *et al.*, 1995) and crop (Barnes *et al.*, 1980) in chickens.

Alternative dietary methods to induced moulting: egg production

Alternative methods of inducing a moult may ameliorate the problems posed by feed withdrawal associated with animal welfare issues and increased *S. enteritidis* infection. Supplementing laying hens' rations with an ingredient which restricts feed intake may be a reliable alternative for reducing body weight and induced moulting. Self-restricting by diet is accomplished by adding drugs or chemicals to a regular diet, or by feeding a diet deficient in one or several essential nutrients, so that the laying hens will voluntarily restrict feed consumption themselves once provided free access to the diet (Lee *et al.*, 1971). Low-calcium (Nevalainen, 1969; Blair and Gilbert, 1973; Gilbert and Blair, 1975;

Hurwitz *et al.*, 1975; Campos and Baião, 1979; Gilbert *et al.*, 1981; Mather *et al.*, 1982), low-sodium (Hughes and Whitehead, 1974; Nesbeth *et al.*, 1974; Whitehead and Shannon 1974; Begin and Johnson, 1976; Dilworth and Day, 1976; Nesbeth *et al.*, 1976a, 1976b; Whitehead and Sharp, 1976; Monsi and Enos, 1977; Herbert and Cerniglia, 1978; Hughes and Whitehead, 1979; Ross and Herrick, 1981; Bird *et al.*, 1982; Ingram *et al.*, 1982; Mather *et al.*, 1982; Said *et al.*, 1984), and high-zinc diets (Scott and Creger, 1976; Herbert and Cerniglia, 1978; Roberson and Francis, 1979; Shippee *et al.*, 1979; McCormick and Cunningham, 1987, Park *et al.*, 2004) have all been examined as possible dietary alternatives. A low-calcium diet less than 0.2 to 0.3% reduced the rate of egg production to less than 5% within 10 to 14 days and in some cases, there was complete cessation of egg laying within 21 days. Rolon *et al.* (1993) found that a low energy, low density, and low calcium diet will cause an egg pause in laying hens when compared with feed withdrawal. A low-sodium diet less than 40 ppm reduced the rate of egg production to less than 5% within 14 to 21 days and in some cases resulted in a complete cessation of egg laying within 4 weeks. A high-zinc diet has received more attention as a potential moulting diet since addition of a trace component is much easier to execute in practical moulting settings than to reproduce a well balanced low calcium and sodium diet (Ruszler, 1998). A diet with 10,000 to 25,000 ppm added as zinc oxide or zinc acetate resulted in cessation of egg production within 5 to 7 days (Shippee *et al.*, 1979).

An alternative dietary approach to feed withdrawal moult induction has been to implement a low energy complete diet that can be fed *ad libitum*. Vermaut *et al.* (1997) reported that a 12% Jojoba supplementation diet was effective for moult induction. Jojaba meal, which is a by-product after the extraction of oil from jojoba seeds, contains approximately 30% crude protein and supplementing feed with defatted jojoba meal inhibits feed intake in chickens (Ngou Ngoupayou *et al.*, 1982; Arnouts *et al.*, 1993; Vermaut *et al.*, 1997). Medvedev *et al.* (2001) found that a high fibre diet consisting of low metabolizable alfalfa in either meal or pellet form resulted in similar ovary size reductions when compared to those of hens undergoing feed withdrawal as well as equivalent egg production and quality.

Alternative methods to induced moulting: *S. enteritidis* colonization

Holt *et al.* (1994) reported that laying hens on alternative moult diets shed less *S. Enteritidis*, showed less susceptibility to an *S. enteritidis* infection and less intestinal inflammation as compared to fasted hens, although the percentage of *S. enteritidis-positive* birds did not differ between moulted groups. Recently, Seo *et al.* (2001) found that feeding wheat middlings, by-products of wheat flour that contain low fibre and higher energy, resulted in cessation of egg production within 7 days, with no increased risk for *S. enteritidis*. Alfalfa moult diets have also been shown to limit *S. enteritidis* infection of laying hens (Kwon *et al.*, 2001). High zinc (10,000 mg/kg) as Zn acetate 10,000 (Moore *et al.*, 2003) and low calcium (80 mg/kg) or low calcium (80 mg/kg)-moderate zinc (110 mg/kg) (Ricke *et al.*, 2003) were very effective for inducing moult and stimulating multiple laying cycles without increasing the risk of *S. enteritidis*. These moult diets not only appear to retain sufficient protective microflora during induced moulting but support sufficient fermentative activity to limit *S. enteritidis* colonization to the same degree as birds on a non-moult layer diet. Availability of such diets would avoid the more drastic influence on the laying hen's gastrointestinal tract microenvironment that feed withdrawal incurs and the subsequent increases in *S. enteritidis* colonization and infection (Durant *et al.* 1999; Ricke, 2003).

Summary and conclusions

Economically in the U.S. induced moulting continues to be a viable managerial option for the egg industry. Complete removal of feed for a period of time has been the traditional approach for inducing moult, but this strategy has come under increasing scrutiny due to physiological and food safety issues. Alternatives to feed withdrawal have been examined historically but are now experiencing renewed interest as the need to replace feed withdrawal has become more of an immediate issue. Development of successful alternative diets has been evaluated on their effectiveness to limit *S. enteritidis* colonization but as animal welfare becomes more of a concern these diets will need to be re-examined for their ability to influence animal behaviour in a positive fashion when compared to feed withdrawal responses. However, to accomplish this will require behaviour indicators that are not only relevant but quantifiable. In addition, physiological bone metabolism and neurological responses may prove to be important parameters to measure and evaluate respective alternative moulting approaches. Several alternative moulting diets have proven to be effective in preventing or at least limiting initial *S. enteritidis* colonization in the laying hen gastrointestinal tract. However, once *S. enteritidis* becomes established in laying hens control measures are fairly limited. Antibacterial approaches such as *Salmonella* specific bacteriophage or bacteriocins may provide some possible avenues for future control strategies in laying hens during moult.

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